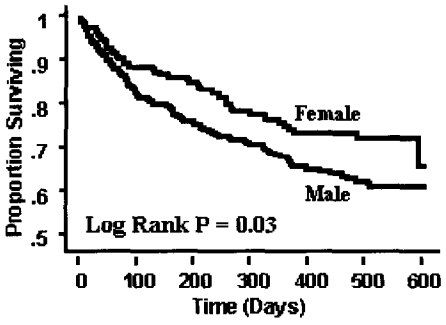
Conclusion: Women admitted with decompensated heart failure appear to have better survival than men after hospital discharge. These results suggest that biological differences in the progression of heart failure are present even with advanced disease.



1169-57 **A Targeted Approach to Reducing Radiation Exposure in the Cardiac Catheterization Laboratory**

Samir V. Germanwala, Venkatraman Srinivasan, Leslie Boltey, Holly James, Alan Gradman, The Western Pennsylvania Hospital, Pittsburgh, PA

Background: Exposure to ionizing radiation is an inevitable consequence of therapeutic procedures performed in the catheterization laboratory. The risk of radiation-induced injury is dose-dependent and numerous injuries secondary to lengthy fluoroscopic times are reported annually to the Food and Drug Administration. In July 2001, we implemented a multifaceted program designed to reduce radiation exposure.
Methods: Physicians and staff were required to demonstrate proficiency with a mandatory radiation-safety curriculum. In addition, fluoroscopic exposure time was documented and tracked *during* each procedure. Operators were informed at ten-minute intervals beginning after 30 minutes of exposure and warned if they were approaching a pre-determined threshold (60 minutes) associated with documented risk of radiation-induced skin injury. To determine the effectiveness of this program, we compared fluoroscopic times in 1,087 patients undergoing interventional procedures during the six-month interval before (n=569) and after (n=518) program implementation.
Results: Average fluoroscopic exposure time decreased from 14.5±3.1 to 9.7±3.7 minutes (p<0.03), a reduction of 31%. Furthermore, there was a 35% reduction in the number of patients sustaining prolonged fluoroscopic exposure (> 40 minutes). There was no statistical difference in baseline characteristics of the two groups or in the percentage of patients in whom factors expected to prolong fluoroscopic exposure time (multi-vessel intervention, intravascular ultrasound, measurement of fractional flow reserve or brachytherapy) were present.
Conclusion : Didactic education and *real time* feedback to physicians during interventional procedures reduced average fluoroscopic exposure times and the number of patients exposed to prolonged irradiation by approximately one-third. Easily replicable, this simple program could significantly reduce the dangers of radiation exposure during interventional cardiology procedures.

1169-58 **Beta Carotene Increases All-Cause Mortality and Cardiovascular Death: A Pooled Analysis of Randomized Trials**

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Background: Beta carotene supplementation has been previously found to be associated with adverse outcomes in patients at high-risk for lung cancer. However, the impact of beta carotene treatment on mortality in patients across the spectrum of cardiovascular risk has not been well-studied. Therefore, we performed a pooled analysis of randomized trials of beta carotene therapy in both low-risk and high-risk patients.
Methods: We pooled the results of four trials which randomized patients to either beta carotene or control therapy. Only trials which included 1000 or more patients were included in the analysis. The dose of beta carotene ranged from 20mg to 50mg.
Results: Four trials, enrolling 90,054 patients fulfilled the inclusion criteria. The pooled all-cause mortality rate was 10.5% in the beta-carotene arm and 9.9% in the control arm. The odds ratio of death for patients treated with beta carotene was 1.07 (1.02-1.12;P=0.003). The rate of death from cardiovascular causes was 4.9% in the beta carotene group and 4.5% in the control therapy group. The odds ratio for cardiovascular death with beta carotene therapy was 1.1 (1.03-1.17; P=0.003). There were no significant differences in rate of cerebrovascular accident between patients treated with beta carotene and those treated with control therapy (4.1% vs. 4.2%, respectively; P=0.59). The Breslow-Day test for homogeneity of the odds ratios was not significant (P=0.26).
Conclusion: Supplementation with beta carotene is associated with a significant increase in all-cause mortality and cardiovascular death in patients at risk for coronary disease. The risk of death from beta carotene therapy appears strongest in smokers. However, a trend towards harm with beta carotene treatment was found even in low-risk patients. The adverse effect of beta carotene treatment on all-cause mortality was found at dosages used in standard preparations of over-the-counter multivitamins. Therefore, the addition of beta carotene to multivitamin preparations should cease and the routine use of these multivitamins by consumers should be discouraged.

Special Topics

POSTER SESSION

1193 Outcomes of Acute Coronary Syndromes

Tuesday, April 01, 2003, Noon-2:00 p.m.
McCormick Place, Hall A
Presentation Hour: Noon-1:00 p.m.

1193-51 **Treatment and Outcomes of Eastern European Patients With Acute Coronary Syndromes in a Multinational Randomized Clinical Trial**

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Background: Registries and clinical trials have offered limited evidence on the translation of non-ST segment elevation acute coronary syndrome (ACS) trial findings into local practices in Eastern Europe (EE). We examined differences in ACS treatment and outcomes between EE and other regions in OPUS TIMI-16, a randomized trial of prolonged oral glycoprotein IIb/IIIa inhibition.
Methods: The OPUS-TIMI 16 trial included 10,288 ACS patients at 753 sites in 28 countries. Of these patients, 1248 (12.1%) came from 62 sites in the EE countries of the Czech Republic, Hungary, Poland, and Russia. Patients received adjuvant treatments at physician discretion. We compared variation in baseline characteristics, treatment, and outcomes of patients in EE and other regions of the world (RW).
Results: We found that EE patients in OPUS-TIMI 16 had more high-risk features at presentation. We identified significant variation between EE and other regions in use of adjuvant therapies during hospitalization. While only 22.5% of EE patients received hypolipidemics versus 39.9% of patients in RW (p<0.001), we observed more extensive use of angiotensin converting enzyme inhibitors (56.3 versus 36.7 (P<0.001)), beta-blockers (65.3 versus 48.62 (P<0.001)), calcium-channel blockers (8.22 versus 3.92 (P<0.001), and nitrates (64.9 versus 26.8 (P<0.001)) in EE. EE patients also underwent fewer percutaneous coronary interventions (PCI) (11.86% versus 29.93% (P<0.001). Patients in EE had worse outcomes at 30 days. After adjustment for baseline characteristics and revascularization rates, EE patients had higher rates of death (hazard ratio (HR) 1.82 (P=0.005)), and myocardial infarction (HR 1.84 (P=0.001)). These trends persisted at 10 months.
Conclusion: Despite similarities in ACS management guidelines, this study revealed disparities in use of hypolipidemic agents and PCI between EE sites and RW in a large multinational clinical trial. Given the higher rates of adjusted mortality and myocardial infarction among EE patients, these findings indicate the need for studies to address the sources of treatment and outcome variation, and strategies for improving access to effective cardiovascular therapies in EE.

1193-52 **Troponin I Elevation Following Percutaneous Coronary Intervention Does Not Predict Future Adverse Outcomes**

J. William Phillips, Habib Samady, Linda Snyder, Robert H. Christenson, Jennifer Gibson, David E. Bruns, Sharon Sayre, Lawrence W. Gimple, Michael Ragosta, Eric R. Powers, Ian J. Sarembock, University of Virginia, Charlottesville, VA

Background: The importance of troponin I (cTnI) elevation following percutaneous coronary interventions (PCI) has not been evaluated as extensively as CK-MB. This study evaluated the prognostic significance of cTnI elevation following PCI on MACE (death, MI, TVR) at three years.
Methods: We prospectively studied 320 consecutive patients without acute MI who underwent successful PCI (mean age 62 +/- 12, males 64%, diabetes 32%, prior MI 43%, CHF 12%, stent use 76%). All patients had blood drawn before and 12-24 hours after PCI for cTnI assay on a Dimension ® RxL. Serial follow-up by phone and questionnaire occurred at six months, one, two and three years.
Results: Periprocedural cTnI elevation occurred in 192/320 (60%) patients using cTnI ≥ 0.1 ng/mL. Using cTnI ≥ 1.5 ng/mL elevation occurred in 70/320 (22%), a rate similar to other studies with CK-MB. Patients with periprocedural elevation of cTnI ≥ 0.1 ng/mL had similar rates of death (9% Versus 10%, p=ns), death and MI (15% Versus 18%, p=ns) or any MACE (33% Versus 32%, p=ns) at 3 years. After controoling for other variables, by multivariable logistic regression analysis, cTnI as a continuous variable after PCI was not a significant predictor of death (χ^2 2.7, p=0.10), death and MI (χ^2 1.9, p=0.17), or death, MI, and TVR (χ^2 0.0, p=0.96) at three years.
Conclusion: After control for common variables, cTnI elevation following PCI does not add prognostic information about future MACE for up to 3 years post-PCI. This may be due to a higher sensitivity of cTnI to detect small amounts of myocardial injury that are not associated with worse long-term outcomes. This has important implications for the design of clinical trials and suggests against the use of cTnI elevation following PCI as a primary endpoint.